Amendment to the Specification

Please insert the following sentence into the first line of the specification:

This application is 371 National Stage filing of PCT/GB2005/000188 filed January 18, 2005, the disclosure of which is hereby incorporated by reference, which claims priority to GB 0403509.3, filed February 17, 2004, the disclosure of which is hereby incorporated by reference.

Please replace the paragraph at lines 1-11 of page 8 with the following:

to one of these compounds, named "galnon" (Wu et al. (2003) Eur. J. Pharmacol. 482 133-137). Galnon equally activates and have agonistic activity to both GALR1 and GALR2. In addition, recent work shows that this compound also activates a number of other GPCR receptors including the neurotensin receptor (abstract Wang et al., Functional activity of galanin peptide analogues. Program No. 960.4 2004 Abstract Viewer/Itinerary Planner. Washington DC: Society for Neuroscience, 2004. Online. (http://sfn.scholarone.com/itin2004/index.html). Thus galnon is not specific in its activation of galanin receptors nor is it a GALR2-specific agonist. The patent application WO02/096934 claims use of galnon in the treatment of pain, epilepsy, but makes not specific claim in relation to the use of such a compound in the treatment of brain injury, trauma or disease.

Please replace the paragraph at lines 15-24 of page 19 with the following:

Organotypic or dispersed primary hippocampal cultures were at various times cultured with or without the addition of the following chemicals: staurosporine (Sigma), L-glutamic acid (Sigma), galanin peptide (Bachem, Merseyside, UK), the high-affinity GALR2-specific agonist AR-M1896 [(Gal(2-11)Trp-Thr-Leu-Asn-Ser-Ala-Gly-Tyr-Leu-Leu-NH₂] (SEQ ID NO: 1) (AstraZeneca, Montreal, Quebec, Canada), amyloid- β (1-42) (A β (1-42)) and the reverse A β (42-1) peptide (American Peptide Company, Sunnyvale, CA 93906). Before use in the experiments below, the A β (1-42) was induced to form fibrils by pre-incubation in culture medium. Specifically, 0.45mg of A β peptide was dissolved in 20 μ l of dimethyl sulfoxide

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(DMSO-Sigma) and diluted to a 100-μM stock solution in medium, which was then incubated with gentle shaking at room temperature for 24 hours.

Please replace the paragraph at lines 16-24 of page 20 with the following:

The standard EAE model of MS was used as previously described (Radu *et al.* (2000) Int. Immunol. 12 1553-60). Mice were immunized subcutaneously in one hind leg with a total of 200 µg of MBP 1-9 (AcASQKRPSQR, (SEQ ID NO: 2) synthesized by Abimed, Langenfeld, Germany), emulsified with complete Freund's adjuvant (Sigma) supplemented with 4 mg/ml *Mycobacterium tuberculosis* strain H37RA (Difco, Detroit, MI). *M. tuberculosis* purified protein derivative (PPD) was obtained from the UK Central Veterinary Laboratory (Weybridge, UK). Mice were scored for symptoms of EAE as follows: 0, no signs; 1, flaccid tail; 2, partial hind limb paralysis and/or impaired righting reflex; 3, full hind limb paralysis; 4,hind limb plus fore limb paralysis; and 5, moribund or dead.